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Foreign Animal Disease Report

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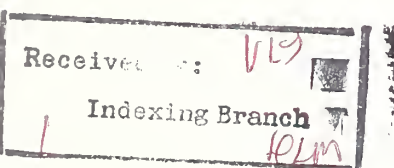
Emergency Programs Activities

Field Investigations. During the first half of Fiscal Year (FY) 1991 (October 1, 1990, to March 31, 1991), veterinarians from the U.S. Department of Agriculture (USDA), Animal and Plant Health Inspection Service (APHIS), Veterinary Services (VS), and State departments of agriculture conducted 97 investigations of suspected foreign animal disease in the United States and Puerto Rico to eliminate the possibility that an exotic disease has entered the United States. These investigations included 57 for vesicular conditions, 10 for swine septicemic conditions, 5 for mucosal conditions, 6 for exotic Newcastle disease, 1 for avian influenza, 2 for encephalitic conditions, and 16 for undesignated conditions. No foreign animal diseases or pests were found.

Exotic Newcastle Disease. On March 29, 1991, a Las Vegas, NV, veterinarian reported that he had examined a client's 2-month-old, yellow-nape Amazon parrot showing tremors and central nervous systems (CNS) signs. The veterinarian suspected Newcastle disease, euthanized the bird, and reported the case to State and Federal animal health officials. A foreign animal disease diagnostician immediately investigated the report and submitted the carcass to the National Veterinary Services Laboratories (NVSL), Ames, IA, where exotic Newcastle disease virus was isolated from the carcass on April 7. Epidemiologic investigations indicate that the bird was purchased March 23, 1991, from a vendor near a swap meet in Spring Valley, CA. Efforts are continuing to locate the vendor and trace other birds that might have been exposed.

In a second incident, a California veterinarian reported several young yellow-nape Amazon parrots exhibiting severe CNS symptoms. He collected laboratory specimens and notified State animal health officials. The owner stated that 60 of 70 young yellow napes had died recently. The California veterinary diagnostic laboratory isolated a Newcastle virus which was characterized as an exotic Newcastle disease virus on April 22, 1991. The 10 yellow napes, 4 pigeons, and 5 doves remaining on the owner's premises were euthanized. All 70 of the parrots reportedly were purchased during the end of March from a street vendor in Venice Beach, CA. Efforts were initiated to locate the vendor and determine if other birds might have been exposed to the disease.

Exotic Newcastle disease has not been reported in commercial poultry in the United States since 1974. Except for 1976, 1978, and 1990, outbreaks have occurred each year since 1972 in pet birds that were illegally brought into the United States. In January, APHIS renewed an intensive effort to alert the public to this disease and



problems associated with smuggled birds. English and Spanish news releases on the outbreaks and factsheets on exotic Newcastle disease and smuggled birds were issued.

Emergency Preparedness. A team met during the week of March 17 in Hyattsville, MD, to finalize plans for an exercise to test APHIS emergency animal disease preparedness.

VS' Resource Management Support staff convened a panel April 2–5, 1991, to revise the Emergency Animal Disease Administrative Guide. Each member was assigned to complete one section of the guide.

A notebook of guidelines for the control and management of African horse sickness was completed, and copies were distributed to appropriate Regional Emergency Animal Disease Eradication Organization members. The APHIS guide for control and management of foot-and-mouth disease will soon be updated.

Action steps of the VS Strategic Plan for emergency disease detection and response have been defined, and the requirements of the plan are being implemented.

(Dr. M.A. Mixson, USDA, APHIS, VS, Emergency Programs, Hyattsville, MD, 301-436-8073)

Foreign Animal Disease Update

This update article summarizes disease surveillance information taken from various sources including bulletins of the Office International des Epizooties (OIE) covering October, November, and December 1990.

OIE received a total of 95 reports describing 593 outbreaks of **foot-and-mouth disease** (FMD) for the last quarter of 1990: 117 from the African countries, 249 from South America, 84 from Asia, and 143 from Europe, the U.S.S.R., and Turkey.

In Africa, Algeria reported a total of 55 outbreaks of FMD type O for July, October, and November 1991. (The Spring 1991 issue reported 53 outbreaks in Algeria for July 1990.) Type O was also the cause of two outbreaks in Egypt during September and nine during November. The outbreak of type O in Morocco that was reported earlier (see 19-1: 4, 1991) occurred near the town of Oujda in northwestern Morocco, along the Algerian border. The 170 sheep involved in the outbreak either died or were slaughtered; 38 new cases appeared during December. Cote-d'Ivoire reported nine outbreaks of FMD type SAT 2 for October and November. Ghana and Togo reported three outbreaks of untyped FMD during November. Togolese veterinary officials reported two possible FMD outbreaks detected February 22, 1991. Seven oxen died, and 25 animals were slaughtered. Diagnosis was based on clinical and postmortem findings. The outbreaks occurred in Mandouri in the Tone Prefecture and Mango in the Oti Prefecture. Benin reported six outbreaks of untyped FMD for March, April, and October. Burkina identified 11 outbreaks of untyped FMD for September and October, and Nigeria reported 2 outbreaks of untyped FMD for May and 2 for October. Chad reported the presence of untyped FMD but no outbreaks during August through November. Sudan reported an outbreak of untyped FMD for October and two for January, and Ethiopia reported four for May. Kenya reported an outbreak of type A in July, one of type O and one of type SAT 2 in August, and one of type SAT 2 in October.

In Europe, Turkey reported 64 outbreaks in September and 79 in October.

In Asia, Jordan submitted reports indicating untyped FMD activity for the first half of 1990. Israel's Ministry of Agriculture reported two outbreaks of FMD in March 1991. The first outbreak occurred in the Golan district, about 7 km from the Israeli–Syrian–Jordanian border, affecting a 2-year-old dairy cow. There was no evidence of spread to other cattle in the herd; all other cattle had been vaccinated and were apparently immune. The other outbreak was from Kefar Zetim in the Kineret district, where 10 dairy cattle were diagnosed positive by the Kimron Laboratory. Both outbreaks were caused by FMD virus type O-1. Control measures included the quarantine and vaccination of all susceptible animals in the affected villages and restriction of animal movements within a radius of 10 km.

Iran reported outbreaks of type O and untyped FMD for April through June 1990 and the presence of type Asia-1 and A without outbreaks during June 1990. Oman identified nine type O FMD outbreaks for September. Pakistan reported two outbreaks of type O for October, and the presence of types O, A, C, and Asia-1 without outbreaks during September through November. Myanmar reported types O, A, and Asia-1 in September and outbreaks of untyped FMD in September and November. Malaysia reported an outbreak of untyped FMD in November. An outbreak of FMD type O was reported from China for July, and Hong Kong identified outbreaks of untyped FMD in September and October.

In South America, Argentina submitted reports for 177 FMD outbreaks for August and September involving cattle, sheep, goats, and swine. Ten of the Argentine outbreaks were attributed to type O, 17 to type A, 2 to type C, and 148 to untyped FMD virus. The Pan American Foot-and-Mouth Disease Center (PANAFTOSA) identified 11 outbreaks of FMD in Bolivia and 21 outbreaks of type O and 1 of type C in Ecuador. During September through November, Colombia identified 32 outbreaks of type A and 2 of type O in cattle. Paraguay reported an outbreak of untyped FMD in bovids during November, and Uruguay reported an outbreak of type C during February.

OIE received 26 reports describing 153 outbreaks of **vesicular stomatitis** (VS) in Mexico and Central and South America. Mexico reported six outbreaks of New Jersey (NJ) type VS for September and October. PANAFTOSA diagnosed 19 outbreaks of VS-NJ in El Salvador during July, 18 in Honduras during August, and 6 in Costa Rica during September 1990.

PANAFTOSA also identified VS-NJ virus as the cause of four Guatemalan outbreaks in July and five Nicaraguan outbreaks in August. Panama reported VS-NJ and Indiana (IN) type VS activity for September, and Colombia reported 52 outbreaks of VS-NJ and 40 of VS-IN for September through November in cattle.

Italy reported two outbreaks of **swine vesicular disease** in February 1991, but no outbreaks of this disease were reported to OIE during the last quarter of 1990.

In Africa, **rinderpest** (RP) was reported from Ethiopia and Kenya. From January through September 1990, Ethiopia reported 5 outbreaks involving 50,590 bovids with 822 deaths. Kenya identified an RP outbreak in Kakuma, along the Ethiopian border.

The December 1990 outbreak involved approximately 10,000 cattle with 20 deaths and 100 new cases at the end of the month. Kenya's last previous outbreak of RP was in May 1989. Rinderpest outbreaks were also reported in Asia from Sri Lanka. The 11 outbreaks from Sri Lanka occurred from July to August 1990 and resulted in 88 cattle dead and 28 slaughtered.

Ethiopia, Ghana, Guinea, and Mauritania reported **peste des petits ruminants** (PPR) activity for the first three quarters of 1990. Cote-d'Ivoire also reported PPR activity September through November. Nigeria identified 13 outbreaks from May to October. The Nigerian outbreaks affected 2,392 sheep and goats and resulted in 68 deaths. In September, Oman identified 8 outbreaks affecting 135 sheep and goats.

In Africa, Guinea reported **contagious bovine pleuropneumonia** (CBP) for January through November 1990. Cote-d'Ivoire reported an outbreak in November affecting 70 cattle and the presence of disease in September and October. Burkina reported that 10 outbreaks in January through September killed 92 cattle, and Benin reported that 10 cattle died in 3 outbreaks during February and 10 died in an outbreak in June 1990. A September CBP outbreak reported from Nigeria involved 222 cattle. Twelve outbreaks were reported from Soba, Sudan, for April and May. Ethiopia reported that 7 CBP outbreaks during the first three quarters of 1990 killed 21 of 30,000 cattle. There were 14 outbreaks of CBP in Kenya, July through November.

In Europe, Portugal reported 164 outbreaks of CBP for July and August 1990, involving 1,088 cattle; 314 were destroyed, and 409 were slaughtered. Spain identified four outbreaks affecting 140 cattle during October and November in the northern Basque region. All affected cattle were destroyed. In October 1990, Italy reported its first case of CBP since the disease was eradicated there in 1899 (see also 19-1: 5, 1991). The first outbreak, in Bergamo Province, affected 548 cattle. All were slaughtered. The disease spread to Mantoue, Come, Cremona, and Cuneo, resulting in 10 more outbreaks in November and December.

In Africa, Egypt reported two outbreaks of **lumpy skin disease** (LSD) in November affecting 33 cattle, and Mauritania reported that the disease was present during January through October 1990, without outbreaks. LSD was also reported to be present without outbreaks during October through December in Cote-d'Ivoire and during November in Chad. Separate outbreaks were reported from Sudan for January, February, August, and September. Ethiopia had several outbreaks from January to September 1990 in which 17 of the 79,600 affected cattle died, and Kenya had 19 outbreaks from July to November. The presence of LSD was reported by Mozambique for October and by South Africa for August through November. The Ministry of Agriculture in Maputo, Mozambique, reported that an outbreak of LSD affecting four free-ranging cattle herds was detected December 24, 1990. The 75 cases occurred in Chiango, Maputo Province. The diagnosis of LSD was based on clinical and laboratory findings. It is reportedly exotic to this area, and introduction is believed to have occurred by migration of herds belonging to refugees. A quarantine has been placed on all affected herds, and a vaccination program has been instituted for animals at risk. A ban has also been placed on the movement of animals and their products between infected and free zones. Four outbreaks of LSD were reported in Swaziland during June 1990 and four in Zimbabwe during November 1990. Madagascar identified 51 outbreaks of LSD with 5 deaths, May through September 1990.

Rift Valley fever was reported from the bordering nations of Malawi and Mozambique. The March outbreak in Malawi killed 10 of 185 affected animals. The disease was present in Mozambique during October without reported outbreaks.

A **bluetongue** outbreak was reported from Mt. Darwin, Zimbabwe, affecting 131 animals. South Africa and the United States reported the presence of bluetongue for August through November and September through December, respectively.

In Africa, Algeria and Mauritania reported **sheep and goat pox** (SGP) during 1990. Algeria identified 37 outbreaks from July through November; Mauritania found SGP throughout the year. Seven outbreaks of SGP in Burkina primarily affected goats and resulted in 59 deaths. Cote-d'Ivoire reported SGP for September, October, and November; and Sudan reported outbreaks for January, February, and August. The bordering nation of Ethiopia submitted a cumulative report for January through September stating that 7 outbreaks of SGP had affected 17,438 sheep and goats.

Turkey has consistently reported the most cases of SGP to the OIE. During September, Turkey reported 143 outbreaks affecting 695,364 sheep (57 dead) and 1,000 goats. A total of 180 outbreaks there during October killed 83 of 794,941 affected sheep.

In the Middle East, Jordan had SGP during the first half of 1990. Iran reported 17 SGP outbreaks for April, 10 for May, and 12 for June. Two outbreaks with 41 cases of SGP were identified in the Golan and Ramla districts of Israel. Seventy-three unvaccinated sheep were affected; 17 died. Quarantine measures were imposed in both areas, and susceptible animals nearby were vaccinated. This was the first incidence of SGP in Israel since February 1986.

In Asia, Pakistan reported SGP during October and November, and Sri Lanka reported an outbreak in the north-central province during January 1991 involving 30 goats.

Reports of **African horse sickness** (AHS) were received from Spain and four African nations during the final quarter of 1990. Spain had its last outbreaks of AHS for the year during October. In the Province of Malaga, 38 outbreaks affected 138 horses, 41 of which were destroyed. Sudan reported continuous AHS activity for January through October. Ethiopia's report for the first three quarters of 1990 identified 7 outbreaks involving 1,112 horses and killing 161. Mozambique and South Africa reported AHS for October and August through October, respectively.

Malawi reported 10 outbreaks of **African swine fever** (ASF) January through October and was the only African country reporting the disease during the last quarter of 1990.

In Europe, Portugal reported single outbreaks of ASF in Serpa and Beja during September and December. A total of 332 swine were affected: 47 died, and 285 were destroyed. Spain reported 23 outbreaks of ASF in September, 7 in October, and 3 in November involving 6,218 swine. In all, 155 died, and the remaining 6,063 affected swine were destroyed. The nine ASF outbreaks reported from Italy were in Nuoro and Cagliari. A total of 149 swine died, and 242 were destroyed.

The OIE received 43 reports of **hog cholera** (HC) for October, November, and December 1990. In the Americas, Mexico reported 6 HC outbreaks: 480 swine were affected in September with 64 deaths and 592 in October with 45 deaths. Uruguay's 3

outbreaks involved 115 swine at Lavalleya, Maldonado, and San Jose during September, October, and November. Argentina reported HC for August and September.

In Asia, the Philippines reported HC August through October, and Sri Lanka reported the disease during October and November. Malaysia had an outbreak of HC involving 150 swine in the peninsular town of Pahang with 8 animals dead and 3 slaughtered. The 22 outbreaks reported from Taiwan affected 17,718 swine with 1,422 dead and 1,024 destroyed. A September outbreak of HC was reported from Hong Kong. South Korea reported 10 outbreaks affecting 365 swine with 222 dead and 143 destroyed.

Outbreaks of HC were reported from Asian and European U.S.S.R. Lithuania had the disease during July 1990, and an outbreak in Orel, where 1,360 animals died and 2,420 were slaughtered, affected approximately 10,000 swine.

In Europe, 70 HC outbreaks were reported at the end of 1990: France (1), Belgium (2), Germany (12), Austria (29), Italy (1), Czechoslovakia (8), and Yugoslavia (17).

Twelve outbreaks of HC, killing 585 swine, were reported from Madagascar for June through September.

The only reports of **Teschen disease** for the last quarter of 1990 came from Madagascar. Madagascar's report described 5 outbreaks each in May, June, and July and 4 in August, killing a total of 295 swine.

A total of 95 reports of **Newcastle disease** (ND) were received in October, November, and December. In Africa, Algeria reported an outbreak in November involving 28,000 birds: 100 died, 8,371 birds were destroyed, and 10,976 were slaughtered for food. An outbreak of ND was reported from Assiut, Egypt, in October. Fifty birds died of ND in Burkina during February. Cote-d'Ivoire recorded ND for September, October, and November. From January to September 1990, Ghana reported 93 outbreaks of ND. The disease centered in Accra and Kumasi and involved 7,734 birds. Government officials estimated that 2,140 birds died. Three separate outbreaks from Nigeria in May, June, and September killed 170 of the 4,330 affected birds. Gabon had ND January through August. Approximately 34,000 birds were affected in outbreaks of ND in Sudan from January to August. Ethiopia reported an outbreak of ND with three deaths during the period January through September 1990. Kenya reported one outbreak of ND in July, one in September, two in October, and three in November. In the Manica district of Mozambique, ND resulted in the death of 6,591 birds and destruction of 3,409. ND was reported from Malawi for January through October and from South Africa for November. The 11 outbreaks reported from Madagascar for July, August, and September resulted in the death of 372 birds.

In Europe, Albania reported three separate outbreaks of ND for August, October, and November. The first two were in Korce and the last in Lushnje. Five ND outbreaks in Turkey during September and October affected 7,905 birds and caused 427 deaths. Yugoslavia reported 11 outbreaks for July, August, and October.

The U.K. Ministry of Agriculture, Fisheries and Food reported two outbreaks of ND from Tyrone and Fermanagh counties, Northern Ireland. Both outbreaks affected egg-laying chickens by causing a sudden drop in egg production. The etiology for the second outbreak was identified as pigeon paramyxovirus-1. Available information points to feed contaminated by infected pigeons as the source of infection. A total of 32,000 birds

were involved in 2,000 cases, with 30 deaths and 32,000 birds destroyed. Control measures included slaughter and onsite burial of all affected birds, establishment of control zones 3 km and 10 km around affected premises, and the investigation of personnel, poultry, and vehicle movements.

In the Middle East, Jordan confirmed the presence ND for January through June, and Iran identified 47 outbreaks for April, May, and June 1990.

In Asia, Sri Lanka reported ND for July through September, and Pakistan, for September through November. Indonesia gave no details regarding outbreaks of ND but did report its presence, January through July 1990. Myanmar's 10 outbreaks during September, October, and November affected 16,361 birds and killed 722. In September, Hong Kong reported one ND outbreak, and South Korea reported two.

In the Americas, Mexico reported an outbreak of ND in September involving 1,000 birds and killing 200 and another outbreak in October affecting 8,000 birds and killing 400.

Velogenic viscerotropic Newcastle disease (VVND) was reported by five countries. The cause of Canada's die-off of wild cormorants, pelicans, and gulls, reported in the spring 1991 issue (19-1: 2-3), was classified as velogenic and mesogenic ND virus.

In Africa, Botswana reported two outbreaks of VVND in September and one in October.

In Asia, Indonesia reported VVND for August through November. Malaysia identified six outbreaks in July, September, and October, involving 10,235 birds (1,665 dead and 3,000 slaughtered).

An outbreak of **viral hemorrhagic disease** of rabbits was reported in mid-April 1991 by Israeli veterinary officials. The outbreak occurred in the Nes Ziyvona Biological Institute in imported laboratory rabbits and spread to other rabbits by contact exposure. All 28 caged rabbits were destroyed.

On February 25, 1991, the European Economic Community's Standing Veterinary Committee placed a 2-month quarantine on farms affected by **mystery swine disease** (MSD). The disease has also been called mystery pig disease and mystery reproductive disease. MSD was first described in Germany in December 1990; by the end of February 1991, it involved 1,000 farms there. The Netherlands reported its first cases in February 1991.

The causative agent of the MSD is unknown. Clinical signs are similar to the MSD first described in the United States in 1987. (See 17-3: 1-2, 1989.) The name "abortus blauw," used in the Netherlands for MSD, derives from the fact that the disease causes abortions, and the ears of aborted fetuses are bluish. The European Economic Community held a conference on MSD at Brussels, Belgium, April 29 and 30, 1991.

(Dr. Peter Fernandez, International Services, APHIS, USDA, Hyattsville, MD 20782, 301-436-8892)

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Wildlife Research Center

The possibility of disease transmission to and from wildlife concerns everyone responsible for protecting livestock and poultry from economically important diseases. While they are working to improve the health of wildlife and wildlife ecosystems, personnel at the National Wildlife Health Research Center—a unit of the U.S. Department of the Interior's Fish and Wildlife Service (FWS)—regularly assist natural

resource managers, biologists, and veterinarians in the investigation of wildlife die-offs. In their efforts to minimize the impact of wildlife diseases, the Center's approximately 55 scientists and support personnel simultaneously perform diagnoses and epidemiologic studies that help to reduce health risks for livestock and poultry. An integrated approach is used to provide solutions to wildlife disease problems. Located in Madison, WI, the Center operates offices and laboratories, including a high-security facility for animal isolation.

The Center's Resource Health Team provides immediate technical assistance to field personnel confronted with wildlife mortality. Team disease specialists provide expertise in wildlife disease control, field investigation, and development of wildlife management strategies to prevent disease introduction. The team provides instruction to field personnel on collection, preservation, and shipment of specimens for laboratory examination. When appropriate, team members travel to the problem area to conduct field investigations and assist local personnel in carrying out wildlife disease-control operations.

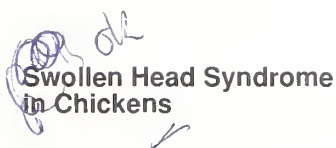
Specimens sent to the Center are processed there. The Center's capabilities include pathology (gross and histopathology), bacteriology, virology, mycology, parasitology, and chemistry. Center disease diagnosticians have been the first to recognize and document several diseases in wildlife.

Center research scientists work to gain new knowledge of disease epizootiology. Other studies include development of methods for improved diagnosis; evaluation of the frequency, geographic distribution, and species affected by various pathogens; evaluation of disease impacts on wildlife population dynamics; and assessment of interactions involving environmental contaminants and infectious agents.

The Center conducts training workshops and seminars on wildlife diseases for Federal or State biologists and other field personnel in Madison and elsewhere. Limited inhouse training is provided to advanced veterinary students, wildlife biologists, and scientists interested in wildlife diseases.

Center activities emphasize species under FWS stewardship, such as migratory and endangered species, and other wildlife found on FWS lands. On a case-by-case basis, the Center's scientists interact with State conservation agencies, other Federal agencies, and the private sector. To request assistance from the Center, contact a member of the Resource Health Team at (608) 271-4640 or FTS 364-5411.

(Dr. Thomas J. Roffe, U.S. Department of the Interior, FWS, National Wildlife Health Research Center, 6006 Schroeder Road, Madison, WI 53711-6223)



Swollen Head Syndrome in Chickens

Occurrence and Epidemiology

Swollen head syndrome (SHS) is an upper respiratory disease of broilers and broiler breeders. Accumulation of fluid under the skin produces the characteristic facial swelling observed in classic cases.

Early descriptions of the condition were reported in South Africa. It was first observed during the mid-1970's, when it was believed to be a secondary problem associated with Newcastle disease. However, increasing incidence led to the recognition of SHS as a separate disease entity. SHS appeared in Europe during 1985 in broilers, layers, and guinea fowl. In 1987, broiler flocks with clinical signs of SHS were reported from Canada.

Etiology and Pathogenesis

Early reports of SHS described rapid spreading in a broiler house during a course of 7 to 14 days. Broilers 4 to 7 weeks old appeared to be most susceptible. In broiler breeders, the disease spread more slowly (over a 2- to 3-week period), affecting hens when they were 24 to 52 weeks old.

Morbidity reportedly ranged from 0 to 50 percent. Depending upon management practices, mortality can reach 20 percent. The condition is aggravated by crowding and poor air quality.

SHS was observed in chickens in Great Britain during the mid-eighties at about the same time that turkey rhinotracheitis (TRT) was occurring in turkeys (see 15-4: 5–7). In many cases, chicken flocks affected with SHS were housed in close proximity to turkey flocks affected with TRT.

Initially, *Escherichia coli* was thought to be the cause of SHS because this microorganism was commonly isolated from affected birds. Some investigators suspected a coronavirus, although other viruses, such as Newcastle disease virus, infectious bursal disease virus, and adenoviruses, also were occasionally isolated. However, the syndrome's temporal association with TRT in turkeys led to the suspicion of a common cause.

In 1987, investigators in the United Kingdom demonstrated TRT antibody in sera from flocks recovering from SHS. Although chickens did not always show clinical signs of SHS, antibodies to the TRT virus were common in chicken flocks. Other workers reproduced the disease by inoculating specific-pathogen-free (SPF) chickens and turkeys with tissue suspensions from SHS-affected chickens. All inoculated SPF turkeys showed the intense rhinotracheitis typical of TRT, and the SPF chickens showed some of the signs observed in SHS flocks. All inoculated birds developed antibodies to TRT virus, and virus was observed by electron microscopy and was isolated from turkey exudates.

TRT virus—a myxoviruslike agent with biological and morphological properties similar to a pneumovirus—appears to be the etiologic agent of SHS in chickens. The virus has been shown to affect turkeys, chickens, geese, guinea fowl, ducks, pheasants, and pigeons.

Clinical Signs

Clinical signs in broilers with SHS progress from an initial transient nasal sneeze or “snick” and a reddening of the conjunctivae to subcutaneous edema of the head. Edema starts around the eyes and spreads to the intermandibular tissues and wattles. The eyelids may close due to swelling and conjunctivitis. Many birds become moribund at this stage. Birds showing severe signs usually die. Birds that survive the acute disease often succumb to secondary infections. The condemnation rate for recovered birds at slaughter is only slightly higher than normal. Morbidity is usually low, but SHS is aggravated by high environmental levels of dust and ammonia.

Broiler breeders and pullets may exhibit CNS signs, including opisthotonos, depression, incoordination, rolling over, and difficulty in regaining normal posture. Egg production may drop by 5 percent and remain at that level for as long as other clinical signs persist. Experimental infection, in the absence of secondary bacterial infections, produces mild clinical signs in the upper respiratory tract.

Lesions

Affected birds show petechiation and red to purple discoloration of the nasal turbinate mucosa. There are yellow, gelatinous fluid and inspissated pus in the subcutis and caseous foci in the air spaces of the cancellous bone of the skull. Secondary infections cause peritonitis, bronchopneumonia, and air sacculitis. An atrophied bursa of Fabricius may also be observed.

Histology and Microbiology

Histologically, many epithelial cells lining the respiratory tract of chickens with SHS are flat and devoid of cilia. There is congestion and aggregation of heterophils and lymphocytes in the subepithelial spaces, and acidophilic cytoplasmic inclusions may be observed in ciliated cells.

Birds with secondary septicemia show a periostitis with purulent inflammation in the air spaces of the cancellous bone of the skull, otitis externa with dense granulocytic infiltrations, and purulent cerebellar meningitis. Bacteriologically, the most consistent finding in many birds is *Escherichia coli* from the heart, liver, peritoneum, and meninges.

Prophylaxis and Treatment

While no commercial vaccine for SHS is available, experimental trials have shown that an attenuated strain of the TRT virus will protect turkeys from TRT. The efficacy of the vaccine under field conditions is currently being investigated.

Secondary bacterial infections may be treated with antibiotics. Affected flocks have responded well to chlortetracycline given in the feed.

Diagnosis

SHS in chickens produces signs that may resemble those of fowl cholera, infectious coryza, or Newcastle disease.

A presumptive diagnosis of SHS can be based on clinical signs and serological evidence. A definitive diagnosis requires isolation and identification of the virus. Samples for laboratory examination should include serum, nasal and ocular swabs, tissues from the trachea and lungs, and scrapings from the nasal sinuses. Swabs, scrapings and tissue samples should be placed in separate containers of transport media for shipment. Samples should be shipped to the National Veterinary Services Laboratory, 1800 Dayton Road, Ames, IA 50010, after reporting the suspected occurrence, as outlined below.

Reporting

SHS has not been reported in the United States. Suspected occurrences of this disease should be immediately reported to Emergency Programs, VS, APHIS, USDA, 301-436-8092 or FTS 436-8092 or to the State animal health authority where SHS is suspected.

(Dr. M.J. David, USDA, APHIS, VS, Import-Export Animals, Hyattsville, MD, 301-436-8590)

24F Focus on Chuzan Disease

An epidemic of congenital anomalies of calves characterized by hydranencephaly-cerebellar hypoplasia (HCH) syndrome took place in the Kyushu district of Japan from November 1985 to April 1986. A new, arthropod-borne virus—Chuzan virus—was suspected of being the etiologic agent of the condition. Antibody to the virus was detected at a high rate in the precolostral serum of affected calves. Chuzan virus, a new member of the palyam subgroup of the genus *Orbivirus*, has been

shown to be the causative agent of the syndrome by virus isolation, seroepidemiological survey, and reproduction of the disease in a newborn calf by experimental inoculation of a pregnant cow with the virus. Sheep, goats, and most likely other domestic and wild ruminants are susceptible to Chuzan virus, although its pathogenicity in these species is unknown.

Chuzan Pathogenesis

Adult cattle are infected subclinically with Chuzan virus. They may show a transient mild leukopenia but otherwise remain normal. One of the characteristics of Chuzan virus is persistent red blood cell-associated viremia. Viremia was observed for several weeks in the presence of specific virus-neutralizing antibody, a phenomenon that also occurs with some of the other orbiviruses, such as bluetongue virus, African horse sickness virus, and others of the palyam subgroup. The reason that the red blood cell-associated virus escapes destruction by the virus-neutralizing antibody is unknown, though change in the three-dimensional structure of the viral surface by adsorption to the red blood cell has been implicated.

Chuzan Signs and Lesions

The most common signs exhibited by affected calves are opisthotonos, epileptic seizures, lack of suckling, and blindness. Calves appear physically normal. Arthrogryposis, which is sometimes seen with bluetongue and Akabane virus infections, has not been reported with Chuzan disease. Hydranencephaly and cerebellar hypoplasia are the primary histologic lesions. Calcium may be seen in the nerve cells or amorphously. Inflammatory reactions, such as cuffing or proliferation of glial cells, are seen less commonly and are generally mild. The inflammatory reactions may be marked during active infection at about the 130th day of gestation but subside by the time the calf is born 150 days later. This phenomenon also occurs in bluetongue virus infections.

Laboratory diagnosis is based on positive serology in conjunction with characteristic histopathologic lesions.

There appears to be no relationship between the age of the cow and the occurrence of HCH in calves. Chuzan virus occurs mainly in beef cows and rarely in dairy cows, though the antibody-positive rates for beef and dairy cattle are very similar. The difference in occurrence rate may be due to a variation in sensitivity of placental tissues to the virus. Age of the fetus at the time of infection appears to be an important factor in the development of clinical disease.

Chuzan Epidemiology

The time of highest prevalence of Chuzan virus in Kagoshima prefecture was estimated to be September 1985 on the basis of seroconversion of numerous sentinel calves in the region. Births of abnormal calves peaked between the end of January and the beginning of February 1986. The time of fetal infection is thus presumed to be about the 130th day of gestation.

The Soo region in the Kagoshima prefecture, Kyushu district, was the site of early reports of abnormal and “dummy” calves born with neurologic signs during November 1985. The affected calves showed no external deformity but had behavioral signs of the HCH syndrome. The number of cases subsequently increased, and the disease spread to adjacent areas, finally affecting cattle in all prefectures in Kyushu. There was a high prevalence in the southern prefectures (Kagoshima, Miyazaki, Kumamoto) and Oita prefecture. Chuzan disease occurred sporadically in the northern prefectures (Fukuoka, Saga, Nagasaki) and occasionally in the Shikoku and Chugoku districts.

Chuzan virus was not seen in the Kyushu district before 1983. Chronological and geographical investigations did, however, indicate the presence of Chuzan virus in Ishigaki Island, the southernmost island of Japan, before 1981. It was detected in sentinel cattle there in 1982, and in the Miyako and Okinawa Islands in 1983. The first two positive cases in Kagoshima prefecture were observed in 1983. Chuzan virus infection in adult cattle and calves with HCH syndrome was detected in the Soo region in Kagoshima prefecture in 1984. Transmission between cattle by vectors was first suspected in 1984, when an orbivirus similar to Chuzan virus was isolated from *Culicoides oxystoma*. The spread of HCH from the primary focus in Kagoshima prefecture to adjacent regions and neighboring prefectures, and the fact that the seroconversion in sentinel calves occurred earlier in Kagoshima prefecture than in other prefectures, suggest that the virus had spread from the original focus. The virus was suspected to have spread to the main islands of Japan through the islands of Okinawa prefecture from southeast Asian countries.

The explosive nature of the outbreak in 1985 and 1986 suggested that Chuzan virus-associated HCH syndrome in calves would appear in the next year in antibody-negative cattle. This did not occur. Neither the virus nor the disease was found in 1986 or 1987. The incidence of disease thus reached peak levels in January and February 1986 and was absent by April 1986.

There has been no recurrence of Chuzan virus-related HCH syndrome since that time. This fact has raised important and as yet unanswered questions about the factors that control disease prevalence. Certainly, the proliferative capabilities of *C. oxystoma* are some of the most important considerations. Unfortunately, many of the conditions that enhance the reproduction of *C. oxystoma* are unknown. Extremes of weather, such as typhoons, may have played a significant role in the dispersion of Chuzan virus vectors. In August 1985, southernmost Kyushu was hit by a typhoon that subsequently moved northward.

Further epidemiological study of the Chuzan virus will necessitate the clarification of the growth and persistence of the virus in cattle and in *Culicoides* spp.

Chuzan Vaccine

Preliminary studies have been undertaken in Japan to develop an inactivated Chuzan virus vaccine. Few vaccinated animals have been tested. Further study is therefore necessary to establish the safety and stability of the vaccine, duration of immunity, and virus-neutralizing antibody titers required to prevent viral infection. Whether inhibition of maternal viremia prevents fetal infection also remains to be determined. With Akabane disease, vaccination is known to prevent viremia and transplacental infection of fetuses.

Laboratory Diagnosis

Laboratory specimens for the isolation of Chuzan disease should include serum for antibody tests and heparinized whole blood collected for virus isolation during seasons of vector activity. Spleen and brain should be collected from calves that have been sacrificed for diagnostic purposes. Half of the brain should be fixed in buffered 10 percent formalin. Specimens should be kept refrigerated but not frozen and delivered to a qualified laboratory without delay.

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